A CASE OF LANDRY'S PARALYSIS WITH RECOVERY.*

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Since the description by Landry in 1859 of an acute ascending paralysis which, beginning in the lower extremities rapidly extends to the trunk and arms and later may involve the respiratory apparatus, thus bringing about death, a great number of cases have been observed both in this country and abroad. All writers are agreed that it is unusual for patients to recover from an attack. However, recently a number of cases of recovery have been recorded, and my excuse for reporting an isolated case is that although the paralysis was complete, involving the bulb and thus producing difficulty in deglutition and dyspnea, recovery took place rapidly and completely. I think that all who listen to this history will agree that the case was a genuine example of acute ascending paralysis, and from the absence of sensory symptoms, there being no history of alcoholism, diphtheria or other infection, will admit that the case could not have been one of multiple neuritis, nor could it have been myelitis, owing to the non-involvement of the bladder and rectum, absence of bedsores, anesthesia and pain; neither could it have been a case of poliomyelitis anterior from the non-existence of muscular wasting or electrical changes.

There has been great confusion in the diagnosis of this disease and many cases of multiple neuritis have undoubtedly been reported as cases of Landry's paralysis with recovery. The diagnosis is not easy from certain aberrant forms of multiple neuritis, those cases, for example, in which there is rapid onset with only slight sensory symptoms, and there are undoubtedly cases of Landry's paralysis in which only the upper and lower extremities are affected and in which the respiratory organs are not involved. It is also difficult in some cases to make the diagnosis between an acute ascending poliomyelitis in an adult in contradistinction to Landry's paralysis, and there is no doubt that some of the cases which have been recorded in the past as spinal paralysis in an adult were cases of Landry's paralysis. I reported one case myself many years ago as one of spinal paralysis of an adult, following Seguin's description, and feel convinced now, with a better understanding of the subject, that the case was one of Landry's paralysis. The patient made a complete recovery.

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I shall not go in detail into the pathology of the disease, as this seems to be still uncertain. The lesions which have been found have been various and there have been no recent observations throwing any additional light on the morbid anatomy. It is certain that many reliable observers have found changes in the spinal cord, indicating vascular and perivascular inflammatory conditions suggestive of the early stages of acute myelitis. Other authors, Bramwell for example, remark that in the majority of cases post-mortem results are entirely negative.

In the case of acute ascending paralysis reported by me in 1891, Dr. Charles W. Burr made a careful examination of the cord and described various changes in the vessels and cells, characteristic of acute myelitis involving the upper part of the cervical cord. Hun and others have also recorded cases of Landry's paralysis in which at post-mortem the cord showed changes similar to those in acute myelitis.

There is one point, however, upon which all recent observers are agreed, and that is that the disease is due to an infection, although no one has pointed out just what the microorganism is. The history and course of every case is suggestive of a definite infection. Although the temperature does not rise to any great extent, nevertheless there is always some febrile reaction, and there is often muscular soreness and general discomfort similar to what is seen in an attack of grippe or other acute infection.

Gordinier, in the *Albany Medical Journal*, 1904, remarks: "I believe, therefore, with Diller that the term Landry's paralysis cannot be dropped, not only because of its long usage, but because by its use we include a group of cases which stand midway between poliomyelitis and multiple neuritis, which cases show definite but slight changes by our newer methods of research confined to the most part to the ventral motor neurones and doubtless due to the circulation in the blood of microbic or metabolic toxins."

The most recent writers, among whom are Knapp, Mills and Spiller hold the view that Landry's paralysis is an "acute parenchymatous degeneration of the peripheral motor neurones of toxic or infectious origin."

Buzzard, *Brain,* 1903, p, 94, in a valuable contribution to the pathology of Landry's paralysis, refers to 25 cases in which bacteria were looked for and not found, either by cultivation or by staining tissues, or by both methods. He points out, however, that changes similar to those met with in the nervous system have been caused experimentally by numerous investigators to be produced by microbic toxins in the absence of microbes themselves. He found 13 cases of Landry's paralysis in which organisms had been demonstrated; and in four of these a diplococcus was present. In a case reported by him he found a micrococcus which stained with Loeffler's methylene blue, in the soft vascular tissue which lies external to the dura mater. In broth inoculations from heart blood, he obtained a culture of a micrococcus indistinguishable from that found in the external part of the dura. The subdural injection of a cultivation of this organism into a rabbit was followed by a rapidly spreading palsy.

Mettler, in the *Journal of the American Medical Asso.*, Vol. 42, p. 1267, quoting Buzzard, Taylor and Clark, concludes that "The pathological changes in Landry's paralysis may be divided into four classes.

"1. Changes in peripheral nerves, indicative of interstitial and parenchymatous neuritis.

"2. Evidence of disease only in spinal nerve roots, and according to Buzzard, the changes are more common in the anterior roots.

"3. Spinal cord involvement, that is, disseminated foci of inflammation, exudates and capillary hemorrhages into the medulla and cord, a swelling of the axis cylinders of the anterolateral tracts and poliomyelitic lesions.

"4. Changes in which all parts of the lower motor neurones, including peripheral nerves, spinal roots and ganglion cells are involved in a scattered and diffuse pathological process."

Rolby reports 7 cases which have been seen in the Medical Clinic at Leipsig in 15 years, and concludes that Landry's paralysis is not a clinical entity but a symptom complex occurring in certain cases of acute polyneuritis, and that cord changes when they occur are due to an ascending neuritis reaching the cord.

It is obvious from the above quotations and numerous others which could be made, that there are still considerable differences of opinion in regard to the nature and pathology of Landry's paralysis, and it is therefore important that all the light possible shall be thrown upon the disease, not only by the report of pathological findings, but also by the recording of cases.

The following patient was seen by me in consultation with Dr. Edward G. Rhoads, who has kindly permitted me to report it:—

S. W. J. Male. Aged 30 years; single. Seen with Dr. Rhoads, May 2, 1904. He has always been temperate as to stimulants and has been a total abstainer for a year. He has smoked moderately. He is employed as a draughtsman in a large machine shop and his hours of work are from 8 A. M. until 5.30 P. M. He has taken much outdoor exercise, has ridden much on horseback and has followed the hounds to a considerable extent. Recently he has been running an automobile every Sunday. He has enjoyed excellent health in every respect and has had no sore throat or any acute disease. On April 24th he ran his automobile about 40 miles. He was not unusually tired nor did he feel chilled when he returned home. The following morning he got up as usual and went to his work, but by evening he felt tired and went to sleep soon after dinner. By bedtime he felt a general stiffness and a slight sense of prickling in his fingers. The day following on rising he felt more stiff, but went to his work, and in the evening went out to dine. He found that he was rather stiff in walking and especially so in going up stairs, and that it was impossible for him to walk very fast. On Wednesday, the third day after the beginning of his symptoms, he went to work as usual and worked all day, but by evening he was still more tired and stiff. On the fourth day he again went to work and found that there was great difficulty in walking. He tried to run to catch a street car and found that it was impossible to do so. By noon of this day he was obliged to give up work and went home. The day following, that is, the fifth day, he sent for Dr. Rhoads. He found that his temperature was normal, and that he was suffering some pain in the calves of his legs. There was no tenderness in the nerve trunks but there was great weakness in both legs and arms. He walked with some difficulty and the grip of his hands was very weak. On Sunday, the seventh day, he was worse in every respect. He fell down while attempting to walk to his bath room and had some difficulty in rising. When seen by me on the following day, I found a well nourished man with good color and healthy appearance. He was sitting in a chair but was unable to rise to his feet without assistance. With one person on either side of him he was raised to his feet and with much support was able to walk across the room. His arms are weak and the grip feeble, but he can perform every movement in a feeble manner. He is unable to cross his legs but can perform other movements feebly. There has been no bladder or rectal weakness; no backache and no girdle pain. He feels tired in his neck when sitting up, but it is not painful. There is a sense of numbness and tenderness in the feet, and his hands feel as if asleep as far as the wrists, but the tactile and pain senses are absolutely preserved and there is no loss of power of localization and no astereognosis. There is complete loss of plantar reflex, knee jerks, Achilles reflex, cremasteric, abdominal and all reflexes in the upper extremities. The only reflex that can be excited is the epigastric. Pupillary reflexes are normal as are all of the facial movements, including movement of the tongue. There is no tenderness on pressure. It was advised that the patient be kept in bed, and aspirin was prescribed.

May 6th. Patient complains of stiffness and numbness in the legs, especially at night. The loss of power has increased materially. He cannot flex his legs, but he can extend them feebly. He can flex and extend the feet and toes but the movement is very weak. These are the only movements in the legs. The grip of the hands is much weaker and he has practically no power to move either arm. There is no change in sensation except that now astereognosis is present. He is unable to determine what is the nature of any object placed in his hand. There is now marked weakness of the facial muscles. He is unable to smile or to make any movements of the mouth, and his face is like a mask. He is unable to raise the upper lip to show the teeth. Protrudes his tongue and closes the eyes well. He has no difficulty in deglutition.

May 8th: Loss of power in the face is greater and now there is only slight movement on left side. In smiling, the left side of the face moves a little, and while he is able to close both eyes the orbicularis is very weak. He is unable to raise the brows. He cannot swallow solids or semi-solids, but can take without difficulty rice which is covered with cream or milk and toast which is very moist. This seems to slip down without causing any trouble. He is sleeping badly. There is more loss of power in his legs and the only movement which he has is a slight power to extend the right leg after it has been flexed and very slight movement of flexion and extension of the feet. This is almost imperceptible. There is no loss of power over the bladder or rectum but he does not know when he has finished urinating, and the bowels are inclined to constipation. The temperature has varied from 98 4-5 to 101 degrees. The pulse is about 80 and respiration 20.

May 10th: Patient is somewhat better as regards the facial movements. Inability to move his lips is not so complete, and the left orbicularis palpebrarum closes a little more strongly. He is still unable to pucker his mouth or to masticate properly. An electrical examination was made on May 9th and showed no change in any of the muscles to the faradic current: all respond promptly and freely. He was very much exhausted after the examination which lasted more than half an hour and this morning he has felt weaker than at any time. The aspirin was now discontinued and bichloride of mercury—1-12 gr. three times a day and strychnia 1-30 gr. four times a day—were ordered.

May 16: Patient has improved in facial movements and the left arm is a little stronger. He is now able to put the hand on top of the head, while three days ago he could not get it as high as his face. The right arm is weaker than the left and the grip weaker. He has less trouble in deglutition, but yesterday was a good deal annoyed by an accumulation of mucus in the mouth, and he is still unable to swallow solids. His appetite is fairly good. Sensation in the feet is not quite as acute as it was, but it is preserved. Bladder and rectum are about the same. From this time on, the patient gradually improved, the power returning first to the arms and later to the legs. By June 1st he was able to sit up and by the end of the month he walked about his room. July 1st he went out to drive and by the latter part of the month he went to the coast of Maine, where he remained the balance of the summer.

He was examined by me again on October 1st, 1904, and was at that time perfectly well and was riding horseback, jumping his horse and following the hounds. About Thanksgiving, 1904, he was taken down with typhoid fever. He did not have a serious attack and his convalescence was about normal. He experienced no more difficulty in walking during this time than anyone else would have. During the third week of the typhoid he had an ischio-rectal abscess which was opened and healed promptly. When seen by me two months later he seemed in perfect health, and weighed 150 pounds. The sensation was normal in feet and hands. Dyn. R. 190 and L. 160 K. JS. were still absent, also the Achilles jerk, but the plantar reflex was present feebly.

I had the opportunity of examining the patient again in the latter part of May, 1906. He was in vigorous health and the knee-jerks were readily obtained. With re-enforcement they were quite active.